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ENVIRONMENTAL DETERMINANTS OF LEAD BURDENS IN CHILDREN

W. A. Galke[1], D. I. Hammer[2], J. E. Keil[3], and S. W. Lawrence[4]

ABSTRACT

Lead burdens in children were studied in relation to exposure to lead from soil and from automobile traffic in Charleston, SC. Preschool aged black children exposed to a variety of soil leads and traffic volumes were recruited in a house-to-house survey. Data regarding soil, traffic, and paint exposure and demographic information also were collected. Soil lead concentrations ranged from 9 to 7,890 ppm with a daily median of 585 ppm. Children's homes faced streets with a daily volume of < 1,000 to 16,000 cars with 51% being in the high traffic recruitment area. Lead paint concentrations varied both within and between homes (0-30 mg/cm^2 in porch railings, 0-51 mg/cm^2 in exterior sidings). Children's blood lead levels ranged from 18 to 77 μg/dl with a mean of 38 μg/dl. Since three representations of traffic volume were available, a separate regression analysis was done for each. Soil lead was always statistically significant while traffic volume and lead paint (exterior siding and door frame) were significant when traffic was represented as high-low. Little influence of demographic or individual characteristics on blood leads was found. These results suggest that measurement of all environmental lead sources must be considered in future lead body burden studies.

RÉSUMÉ

La plombé mie d'enfants de Charleston (Caroline du S.) a été étudiée en rapport avec l'exposition au plomb provenant du sol et des gaz d'échappement de voitures. De jeunes noirs d'âge scolaire, exposés à diverses concentrations de plomb dans le sol et dans l'air, ont été recrutés par porte-à-porte. Des données sur l'exposition au sol, à la circulation automobile et à la peinture, ainsi que les renseignements démographiques ont été recueillis. Les concentrations du plomb dans le sol variaient de 9 à 7890 ppm

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(médiane quotidienne: 585 ppm). Les maisons où habitaient les enfants bordaient des rues où la circulation quotidienne s'établissait entre 1000 et 16 000 voitures; 51% des maisons étaient situées dans un secteur à circulation intense. Les concentrations de plomb dans la peinture variaient pour chaque maison et d'une maison à l'autre (0-30 mg/cm² dans le cas des balustrades et des rampes, 0-51 mg/cm² dans celui du revêtement extérieur). Le plomb sanguin variait chez les enfants de 18 à 77 μg/dl, autour d'une moyenne de 38 μg/dl. Trois représentations différentes du volume de la circulation étant disponible, on a effectué une analyse de régression pour chacune d'elle. La concentration du plomb dans le sol était toujours significative tandis que le volume de la circulation et la concentration de plomb dans la peinture du revêtement extérieur et des cadres de portes étaient significatifs lorsque la circulation était catégorisée "importante" ou "faible." On a trouvé peu d'influence des caractéristiques démographiques ou individuelles sur le plomb sanguin. Les résultats font penser à envisager le dosage de toutes les sources de plomb dans l'environnement, dans le cadre d'études ultérieures de la plombémie chez l'homme.

INTRODUCTION

Good evidence exists linking adverse health effects and increased lead body burdens. Less evidence exists relating increased body burdens with exposure to specific environmental sources of lead. Environmental lead sources of major concern are paint in residential dwellings, and airborne and dustfall emissions, primarily from mobile sources and soil.

Whereas data from many urban lead screening programs have strongly implicated lead paint as a primary source of increased lead burdens in children, quantification of a specific dose-response relationship for lead paint exposures has not been achieved (Lin-Fu 1972; National Academy of Sciences 1972). Furthermore, the earlier studies virtually always considered only paint as a source of environmental lead. Dustfall frequently contains large concentrations of lead and only recently was dust, as well as air, hypothesized as a major source of lead in children with increased lead body burdens (Hammer *et al.* 1972).

Dose-response curves for both epidemiologic (Goldsmith and Hexter 1967) and experimental (Knelson *et al.* 1972) studies of exposure to atmospheric lead have been derived. Studies of exposure to automobile traffic, a major source of atmospheric lead, have not yet reached that level of refinement (Thomas *et al.* 1967; Daines *et al.* 1972; Caprio *et al.* 1974). Existing data suggest a relationship in adults with but a small increase in body burdens (Thomas *et al.* 1967; Daines *et al.* 1972). Children are thought to be at even higher risk of effects than adults, although less data are available. A retrospective study of children from a lead screening program in Newark, New

Jersey showed a qualitative association of traffic exposure with blood lead level (Caprio *et al.* 1974).

Although much speculation has occurred in the literature linking soil lead exposures and lead burdens in children, few studies have actually tested for the association of them. One of these, a retrospective study conducted in Charleston, South Carolina, associated lead poisoning cases in children with elevated soil lead levels (Fairey and Gray 1970). A British report covering two studies found no association between children's blood lead levels and soil lead exposures in one study, but found an association in the other (Barltrop *et al.* 1974). Results from Barltrop's second study in which the association was found, must be considered tentative in that actual soil lead levels for individual children are not available as yet. Limited soil lead data from this study suggested that soil lead levels were much higher than those in the first.

Since evidence links lead in paint, air and soil with increased lead body burdens, studies of any of these single sources should include measurement of the others. The primary hypotheses in this study were (1) blood lead levels are positively related to soil lead levels and, (2) blood lead levels are positively related to automobile traffic independent of soil lead exposure.

METHODS

Lead exposures were assumed to occur at the children's homes. Data were collected to validate this assumption as well as to measure the environmental levels of lead occurring there. Available information on population characteristics (1970 Census), soil lead levels (Fairey and Gray 1970), and automobile traffic densities in Charleston (City of Charleston) were utilized to define areas of recruitment. A series of overlay maps showing soil lead contours, traffic volume of major arteries, and census blocks of 50% or more black population were prepared. From these maps, four recruitment areas of contiguous city blocks with a primarily black population were established: high traffic-high soil lead; high traffic-low soil lead; low traffic-high soil lead: and low traffic-low soil lead. Black pre-school children from these areas were recruited by interviewers in a house-to-house survey during the summer of 1973.

For each participant, a soil sample, a venous blood sample, five *in situ* lead paint measurements, traffic density data, and a questionnaire were obtained. The soil sample was collected from the child's chief play location; the blood sample was collected by venipuncture. Soil and blood lead concentrations were determined by atomic absorption spectrophotometry.

Hematocrit levels and glucose-6-phosphate dehydrogenase (G6PD) enzyme activity were also determined. G6PD enzyme activity was measured by the method of Gadsden. (Gadsden and Cannon 1964).

Exposure to lead from painted surfaces involves not only the absolute lead concentration of the paint but also the quality of the painted surface; flaking paint being easier for the child to ingest than intact surfaces. Paint lead levels were determined in the following five surfaces by x-ray fluorimetry: porch railing, exterior siding, door frame, window sill, and interior wall. The intactness of these same surfaces was assessed by the home interviewer.

As with exposure to leaded paint, exposure to automobile traffic has two components: distance of the home from the road, and the quantity of traffic to which the subject is exposed. Distance of the home from the facing street was measured by the home interviewers. Originally, the only measure of traffic volume was the high or low designation of the recruitment area; then, two additional estimates of traffic volume were derived after recruitment of children was accomplished: daily traffic volume on the street facing the home, and daily traffic volume within a 76 meter (250 ft.) radius of the home.

A questionnaire was administered to the child's parent(s) or guardian(s) regarding information on the family's socioeconomic characteristics, possible other lead exposures, the child's or family's habits which might modify their exposure, and the child's medical history for lead poisoning. Demographic information available on a family basis included the education level of the head of the household, the number of rooms in the residence, and the number of residents in the dwelling. Individual characteristics of importance included history of lead poisoning, presence of pica, and location of the child's primary play site.

Only children with both a blood and soil lead measurement were included for statistical analysis. One hundred ninety-four children from 170 families were recruited for study. Of these, less than 4% of the children were excluded because either a soil or blood lead measurement was not available (7/194). The 187 included children were distributed among 164 families. Descriptive statistics including the mean, median, range and various percentiles were used to describe the lead environment of each child as well as the demographic characteristics of the study children. Correlation and multiple regression analyses were used to describe the interrelationships of soil lead, traffic volume, and lead paint. Regression analyses were made with soil lead as the dependent variable; independent variables were the lead values of the five painted surfaces and a traffic exposure variable. A separate analysis was completed for each of the traffic volume representations.

Statistical analysis of the study hypotheses was accomplished through multiple regression analysis. Blood lead level, not adjusted for hematocrit differences, was the dependent variable, while automobile traffic, soil lead, paint lead, demographic characteristics and individual characteristics were the independent variables. Separate analyses were again made for each one of the 3 traffic volume representations.

RESULTS

Exposure to lead for the 164 families comprising the study population varied within each of its three environmental sources: paint, atmosphere, and soil. Levels of lead in paint varied between homes and within homes (Table 1). Porch railing and interior wall paint lead were similar with medians of 1.0 and 1.3 mg/cm^2, respectively, while medians for exterior siding, window sill, and door frame lead paints ranged from 2.1 to 3.4 mg/cm^2 (90th percentile and maximum values followed a similar pattern). Although all painted surfaces had a zero minimum lead content, the maximum levels varied from about 30 mg/cm^2 in the porch railing to about 51 mg/cm^2 in the exterior siding.

Interior wall paint lead showed little correlation with the other surfaces; this is in contrast to the other four surfaces which were consistently related to one another (Table 2). Door frame and window sill paint lead concentrations were most closely related and an intermediate relationship existed between exterior siding lead and both window sill and door frame lead.

Painted surfaces also varied in quality with about 45% of the homes having flaking paint on at least one surface. Thirteen percent of the homes had

TABLE 1

Lead Concentrations in Painted Surfaces of Family Dwellings.

Painted Surface	Lead Concentration, mg/cm^2		
	Median	90th Percentile	Maximum
Porch Railing	1.0	7.8	30.4
Exterior Siding	2.4	14.7	50.6
Window Sill	2.1	12.3	42.8
Door Frame	3.4	14.4	43.5
Interior Wall	1.3	6.6	36.3

paint chips present in the soil surrounding them. Painted surface quality was not uniform within a home for the five surfaces sampled. Only eight percent of the homes had all their painted surfaces in a flaking condition, whereas 44 percent had all their painted surfaces in a "sound" condition (Table 3). Flaking in the specific surfaces ranged from a low of 19% in door frames to a high of 41% in porch railings (Table 4). Exterior sidings, window sills, and interior walls were intermediate with about 30% of each exhibiting flaking paint. Lead paint content and quality of the surface were related, in that poorer quality surfaces always had higher mean lead levels (Table 5).

Homes of the study population were a median of 9.1 m from the road with a range from 1.2 to 65.8 m. Fifty-one percent of the homes were located in the high traffic exposure recruitment areas. Daily traffic volume on the facing street and daily traffic volume within a 76 m (250 foot) radius of the home were both higher in the high traffic recruitment areas than in the low traffic recruitment areas (Table 6), thus indicating that as a crude measure of traffic exposure, residential location was acceptable. Facing street traffic ranged from 100 to 15,575 cars daily.

Soil lead levels varied greatly, with a range from 9 μg/g to 7890 μg/g (Table 7). The distribution of soil lead levels was positively skewed with a median of 585 μg/g.

Paint lead levels and automobile traffic volume varied with soil lead concentrations. Paint lead levels in all surfaces were higher in the high soil exposure group than in the low exposure one (Table 8). Sixty-one percent of the homes in the high soil group were in the "high" traffic recruitment areas. Despite this, the picture presented by the actual traffic density measurements

TABLE 2

Relationships Among the Paint Lead Levels of the Painted Surfaces.
(Correlation Coefficients)

Painted Surface	Porch Railing	Exterior Siding	Window Sill	Door Frame	Interior Wall
Porch Railing	1.000	–	–	–	–
Exterior Siding	0.331[a]	1.000	–	–	–
Window Sill	0.314[a]	0.426[a]	1.000	–	–
Door Frame	0.178[b]	0.463[a]	0.561[a]	1.000	–
Interior Wall	0.063	0.239[a]	0.189[a]	0.272[a]	1.000

Sample sizes ranged from 153 to 159.
[a] $p \leq 0.01$
[b] $p \leq 0.05$

TABLE 3

Categorization of Family Dwellings by Quality of Combinations of Painted Surfaces.

Number of Painted Surfaces Present	Quality of Painted Surface			Number of Homes*
	Good	Fair	Poor	
Five Surfaces Present and Painted	28.4%	58.0%	13.6%	81
Four Surfaces Present and Painted	51.3%	48.7%	0.0%	39
Three Surfaces Present and Painted	74.2%	22.6%	3.2%	31
Total	43.7%	48.3%	8.0%	151

*Excludes three homes from which no data are available and 10 homes from which data from at least one surface is missing.

Definitions of Terms of Quality for Combinations of Painted Surfaces

GOOD – A home in which all painted surfaces are in sound condition.
FAIR – A home in which some but not all painted surfaces are in flaking condition.
POOR – A home in which all painted surfaces are in flaking condition.

TABLE 4

Categorization of Family Dwellings by Quality of Individual Painted Surfaces.

Painted Surface	Quality of Surface		All Dwellings*
	Sound	Flaking	
Porch Railing	60%	40%	102
Exterior Siding	73%	27%	108
Window Sill	69%	31%	161
Door Frame	81%	19%	161
Interior Wall	69%	31%	161

*Percentages based only on surfaces present.

TABLE 5

Lead in Paint by Quality of Painted Surface.

Quality of Painted Surface	Mean Lead Paint Levels (mg/cm^2)				
	Porch Railing	Exterior Siding	Window Sill	Door Frame	Interior Wall
Sound	2.9	7.6	3.5	6.1	2.5
Flaking	6.4	11.1	5.1	6.7	2.9

TABLE 6

Traffic Volume by Traffic Exposure Area.

Traffic Exposure Classification	Mean Traffic Volume (cars/day)	
	Facing	Within 76 m (250 ft.)
Low (83)*	951	1677
High (80)	2446	9637

*Number of houses.

TABLE 7

Soil Lead Levels at Family Dwellings.

Statistic	Soil Lead, ppm*		
	Low Soil	High Soil	All Dwellings
Median	173	1400	585
75th Percentile	351	2090	1400
Range	9-584	586-7890	9-7890

*High or low designation is based on overall median of 585 ppm.

indicated that similar ranges of exposures were experienced in both the high and low soil exposure groups. Although the range of traffic densities was similar in the two groups, the distribution of densities was not (Table 9). Higher traffic volumes were most frequent in the high soil exposure groups.

TABLE 8

Levels of Lead in Painted Surfaces (mg/cm^2) by Soil Lead Exposure.

Painted Surface	Soil Lead Exposure, ppm	Paint Lead Content, mg/cm^2		
		Median	90th Percentile	Maximum
Porch	< 585	0.0	4.1	25.4
Railing	≥ 585	1.2	9.9	30.4
Exterior	< 585	0.0	13.9	50.6
Siding	≥ 585	3.7	19.4	48.9
Window	< 585	1.7	5.6	27.9
Sill	≥ 585	2.5	15.2	42.8
Door	< 585	4.0	13.2	28.0
Frame	≥ 585	3.1	18.3	43.5
Interior	< 585	1.0	8.3	19.9
Wall	≥ 585	1.4	5.9	36.3

Results of the multiple regression analysis of the interrelationships of soil lead with paint and traffic indicated that soil lead was an independent measure of lead exposure in Charleston. In none of the 3 separate analyses for each traffic exposure representation was a significant relation obtained, and the percent variation explained in soil lead was always less than 30%.

Regressing soil lead levels against each paint lead variable and traffic volume variable independently found significant relations between soil lead and exterior siding paint, window sill paint, and traffic (as a dichotomous variable). Still, none of these relationships explained much of the soil lead variability.

Comparisons between the demographic characteristics of families and characteristics of individuals were made for children exposed to soil lead concentrations below the median value and those exposed to more than this value. Family characteristics were remarkably similar for the two soil exposure groups (Table 10). Less than half of the families were headed by someone with a high school or greater education and only 6% of the heads of households had a college education. There was a median of 5 rooms per home with 55% of the homes having at least five rooms. Median family size was five, with two thirds of the families at least that large.

Likewise, individual characteristics were similar across both soil exposure groups. No difference in the age-sex distribution of the soil exposure

TABLE 9

Daily Traffic Volume by Soil Lead Exposure.

Soil Lead Classification	Daily Traffic Volume (cars/day)			
	Facing Street		Within 76 m. (250 feet)	
	Median	75th Percentile (Range)	Median	75th Percentile (Range)
< 585 ppm	100	1000 (100-12,275)	1100	8875 (100-35,300)
≥ 585 ppm	100	2325 (100-15,575)	3200	8550 (100-35,400)
All Houses	100	1085 (100-15,575)	1100	8750 (100-35,400)

TABLE 10

Descriptive Characteristics of Families.

Soil Lead Category	Head of Household with High School or Greater Education	Number of Residents		Number of Rooms	
		Median	≥ 5 residents	Median	≥ 5 rooms
Low (< 585 ppm)	45.1%	5	67.1%	5	53.7%
High (≥ 585 ppm)	43.9%	6	68.3%	5	56.1%
All Families	44.5%	5	67.7%	5	54.9%

groups was noted. Individual study children were evenly divided by sex and ranged in age from 0 to 5 years, being concentrated in the 1, 2, and 3 year age groups (Table 11). History of lead poisoning, prevalence of pica, and whether the child's chief play location was his home, likewise were quite similar (Table 12). About three percent of the children had a history of lead poisoning, less than 45% of the children exhibited pica, and 90% of the children played primarily at home. Prevalence of pica varied by sex and age (Table 13). Male children had a higher prevalence than female children and the prevalence was higher in the one to three year old children, as expected. Insufficient positive response was obtained for use of homemade pottery and home gardens to warrant consideration of those variables.

TABLE 11

Number of Study Participants by Age and Sex.

Soil Lead Category	Sex	Age in Months					Total
		1-12	13-24	25-36	37-48	49-60	
Low (< 585 ppm)	Female	1	18	15	9	0	43
	Male	6	10	16	13	0	45
High (≥ 585 ppm)	Female	2	11	17	13	0	43
	Male	1	20	16	13	1	51
All Children		10	59	64	48	1	182*

*Five of the children were missing information on age or sex.

TABLE 12

Characteristics of Study Children.

Soil Lead Category	Yes, History of Lead Poisoning	Yes, Child Does Exhibit Pica	Yes, Child Does Play Mainly at Home
Low (93)*	2.1%	44.1%	90.3%
High (94)	3.2%	43.6%	90.3%
All Children	2.7%	43.9%	90.3%

*Number of children, (< 1% nonresponse on these questions).

TABLE 13

Prevalence of Pica in Children by Age and Sex.

	Sex	
Age (years)	Female Percent (n)	Male Percent (n)
0-1	33.3 (1/3)	28.6 (2/7)
1-2	55.2 (16/29)	50.0 (15/30)
2-3	31.3 (10/32)	65.6 (21/32)
3-4	31.8 (7/22)	34.6 (9/26)
4-5	–	100.00 (1/1)
All Ages	39.5 (34/86)	51.1 (48/94)

Biologic variables were not as consistently similar across soil exposure categories. Although no difference was noted in hematocrit, the same was not true for G6PD deficiency. Both the mean and median hematocrit for the study children were 34%, and the range of values was from 25 to 42. No differences were noted for either age or sex in hematocrit levels (Table 14). For both sexes, a higher prevalence of enzyme deficiency (defined as ≤ 25 units) occurred in the more highly exposed children. Although the greatest absolute increase in prevalence across the soil exposure groups was in males, the greatest relative increase occurred in females (Table 15). As expected, males exhibited a much larger prevalence (16.8%) of G6PD deficiency than females (3.5%).

Blood lead levels ranged from 18 to 77 μg/dl with a mean of 36 and a median of 38. Blood leads did not vary by age or sex (Table 16). G6PD

TABLE 14

Mean Hematocrit by Sex and Age.

Age in Years	Sex	
	Female	Male
0-1	32.0%	32.6%
1-2	33.6%	33.2%
2-3	33.3%	33.8%
3-4	34.2%	35.0%
4-5	–	37.0%
All	34.3%	33.9%

TABLE 15

Prevalence of Glucose-6-Phosphate Dehydrogenase Enzyme Deficiency in Study Children by Soil Lead Exposure and Sex.

Soil Lead Exposure (ppm)	G6PD Enzyme Deficiency*	
	Female	Male
Low (< 585 ppm)	2.3%	13.0%
High (≥ 585 ppm)	4.9%	20.4%
All Children	3.5%	16.8%

*Enzyme deficiency is defined as 25 units or less by the method of Gadsden.

TABLE 16

Blood Lead Levels (μg/dl) by Age and Sex.

Age (Years)	Sex		Both Sexes
	Female	Male	
0-1	36.9	31.6	33.7
1-2	35.9	40.6	38.3
2-3	38.6	36.4	37.5
3-4	35.6	40.4	38.2
4-5	–	45.0*	45.0
All Ages	36.9	38.5	37.7

*Only one observation.

deficiency was shown not to affect blood lead levels of children of both sexes given similar exposures (Table 17). How blood lead concentrations varied by exposure to soil lead, autmobile traffic (as measured by residence in recruitment areas) and individual surface paint lead exposures are displayed in Tables 18-23. Blood leads varied with soil lead, automobile traffic, porch railing lead, and exterior siding lead.

Separate analyses made for the three automobile traffic exposure representations resulted in an interesting pattern appearing for that variable. In all three analyses soil lead was significantly positively related to blood lead levels, while traffic exposure and exposure to leaded paint were not. Only in

TABLE 17

Blood Lead Levels by Soil Lead Exposures, G6PD Deficiency, and Sex.

	Blood Lead Concentrations, μg/dl (Mean ± Std. Dev.)			
	Females		Males	
Soil Lead Exposure	Deficient	Not Deficient	Deficient	Not Deficient
Low	37 ± –	33 ± 8	37 ± 9	37 ± 9
High	33 ± 6	41 ± 13	37 ± 9	41 ± 9
All	34 ± 5	37 ± 12	37 ± 8	39 ± 9

*Enzyme deficiency is defined as 25 units or less by the method of Gadsden.

TABLE 18

Blood Lead Levels by Soil Lead and Automobile Traffic Exposures.

	Blood Lead Concentrations, μg/dl (Mean ± Std. Dev.)		
	Automobile Traffic Exposure		
Soil Lead Exposure	Low	High	All Traffic Exposures
Low	32 ± 7	41 ± 8	35 ± 8
High	36 ± 10	43 ± 11	41 ± 11
All Soil Lead Exposures	34 ± 9	42 ± 10	38 ± 10

TABLE 19

Blood Lead Levels by Soil Lead and Porch Railing Paint Lead Exposures.

	Blood Lead Concentrations, μg/dl (Mean ± Std. Dev.)		
	Lead in Porch Railing Paint (mg/cm^2)		
Soil Lead Exposure	Low (< 1.0)	High (≥ 1.0)	All Porch Railing Paint Lead Exposures
Low	33 ± 8	38 ± 8	35 ± 8
High	40 ± 12	41 ± 11	41 ± 11
All Soil Lead Exposures	36 ± 11	40 ± 10	38 ± 10

TABLE 20

Blood Lead Levels by Soil Lead and Exterior Siding Paint Lead Exposures.

	Blood Lead Concentrations, μg/dl (Mean ± Std. Dev.)		
	Lead in Exterior Siding Paint (mg/cm^2)		
Soil Lead Exposure	Low (< 2.4)	High (≥ 2.4)	All Exterior Siding Paint Lead Exposures
Low	33 ± 7	38 ± 9	35 ± 8
High	39 ± 11	42 ± 11	41 ± 11
All Soil Lead Exposures	35 ± 9	40 ± 10	38 ± 10

the recruitment area model were the latter two exposures significant. Traffic exposure was highly significant when represented as a dichotomous variable, that is residence in the "low" or "high" traffic recruitment areas, but was not significant in either of the other representations. Significance levels increased from traffic on the facing street through traffic within a 76 m radius to the traffic recruitment area. In the model using traffic recruitment area, both exterior siding and door frame paint were significant but exterior siding was only marginally significant ($.05 < p < .10$). Door frame was even less signifi-

TABLE 21

Blood Lead Levels by Soil Lead and Window Sill Paint Lead Exposures.

	Blood Lead Concentrations, μg/dl (Mean ± Std. Dev.)		
	Lead in Window Sill Paint (mg/cm^2)		
Soil Lead Exposure	Low (< 3.4)	High (≥ 3.4)	All Window Sill Paint Lead Exposures
Low	35 ± 9	35 ± 8	35 ± 8
High	41 ± 11	41 ± 11	41 ± 11
All Soil Lead Exposures	37 ± 10	38 ± 10	38 ± 10

TABLE 22

Blood Lead Levels by Soil Lead and Door Frame Paint Lead Exposures.

	Blood Lead Concentrations, μg/dl (Mean ± Std. Dev.)		
	Lead in Door Frame Paint (mg/cm^2)		
Soil Lead Exposure	Low (< 3.4)	High (≥ 3.4)	All Door Frame Paint Lead Exposures
Low	34 ± 9	36 ± 8	35 ± 8
High	40 ± 10	42 ± 13	41 ± 11
All Soil Lead Exposures	37 ± 10	39 ± 11	38 ± 10

cant in the other two traffic volume regression models. Little influence of demographic or individual characteristics was noted. Presence of pica in a child was marginally significant ($.05 < p < .10$) in all three analyses. History of lead poisoning was significant in the recruitment area model but not in the other two. All three regression models explained significant proportions of the blood lead variations, with the traffic recruitment area model explaining the largest amount of variation.

TABLE 23

Blood Lead Levels by Soil Lead and Interior Wall Paint Lead Exposures.

	Blood Lead Concentrations, μg/dl (Mean ± Std. Dev.)		
	Lead in Interior Wall Paint (mg/cm^2)		
Soil Lead Exposure	Low (< 1.3)	High (≥ 1.3)	All Interior Wall Paint Lead Exposures
Low	33 ± 7	37 ± 9	35 ± 8
High	40 ± 10	42 ± 12	41 ± 11
All Soil Lead Exposures	36 ± 9	40 ± 11	38 ± 11

DISCUSSION

Children were recruited from four areas of Charleston, South Carolina chosen for presumed soil and traffic lead exposures. These exposures, as well as exposure to leaded paint, were found to vary among the children in each area. Children's blood lead levels were found to be positively related to all three environmental exposures of lead: soil, traffic, and paint. Therefore, the hypothesis that blood lead levels are related to soil exposure was confirmed. Furthermore, the second hypothesis, that blood lead levels are related to exposure to automobile traffic independent of soil exposure was also confirmed. Exterior siding paint and, to a much lesser degree, door frame paint were related to blood lead levels. Only one traffic volume representation was significant, that of residence in a traffic recruitment area. Significance levels of traffic volume varied directly with the size of the geographic area covered by the representation. Relatively little influence of demographic and individual characteristics was noted on blood lead levels. Pica was suggestive and history of lead poisoning slightly less so.

Soil lead levels found in this study, ranging from 9 to 7890 μg/g, agreed with levels found previously in Charleston by Fairey and Gray (1970) whose data ranged from 1 to 12,000 μg/g. Data from other geographic locations indicate that soil lead levels in Charleston are higher than those found in most other areas of the country (National Academy of Science 1972).

Traffic volumes found in Charleston on non-expressway thoroughfares are quite common in the United States. In fact, most studies relating blood leads to traffic exposure have sampled much higher traffic densities from either expressways or very heavily travelled urban thoroughfares (Thomas *et al.* 1967; Daines *et al.* 1972; Caprio *et al.* 1974).

Only limited data are available to compare paint lead concentrations in this study with paint lead concentrations elsewhere. A comparison between lead levels found in homes located in high lead poisoning prevalence areas of New York City and those in Charleston is presented in Table 24. It is readily apparent that using 1.0 mg/cm^2 as a criterion the Charleston and New York homes were similar, but the results of the 5.0 mg/cm^2 criterion indicate New York City paint lead exposures were higher. A similar comparison can be made with data from Cincinnati, Ohio (Table 25). In this case, mean lead paint exposures were less in Charleston although maximum exposures were greater. Therefore, the paint lead exposures in this study do not seem to be abnormally high or low.

In summary, then, children in this study were exposed to traffic and paint exposures similar to those of other urban children, but may have been exposed to somewhat higher soil leads than commonly experienced. If so, the increased soil lead exposure may be reflected in the blood lead levels found in Charleston. In this study, 36% of the children had blood leads over 40 μg/dl. This percentage is somewhat higher than the results obtained from a survey of 27 lead screening clinics throughout the Nation (Simpson *et al.* 1973). In that survey, 8 to 33% of all children screened had lead levels as high as 40 μg/dl.

TABLE 24

Comparison of Paint Lead Levels in New York City[a] and Charleston.

	% of Surfaces with Paint Lead			
	> 1.0 μg/cm^2		> 5.0 μg/cm^2	
Surface	NYC	Charleston	NYC	Charleston
Rooms	87.5	–	56.4	–
Walls	58.5	55.3	32.7	16.3
Door Frame	62.7	86.8	42.2	37.1
Window Frame	69.8	71.1	44.0	17.6

[a]Laurer, G. R. *et al.* (1973).

TABLE 25

Comparison of Paint Lead Levels in Family Dwellings in Cincinnati[a] and Charleston.

Surface	Mean Paint Lead (mg/cm^2)		Range	
	Cincinnati[b]	Charleston	Cincinnati	Charleston
Exterior Siding	11.1	5.8	1.0-30.8	0.0-50.6
Door Frame	10.6	6.2	1.7-26.7	0.0-43.5
Interior Wall	5.1	2.7	0.5-21.2	0.0-36.3

[a]Reece, R. M. *et al.* (1972).
[b]Sample sizes for Cincinnati ranged from 7-62.

About 2% of our children had levels above 60 μg/dl compared to less than 1% in the previously mentioned survey.

The association of all three environmental sources with increased lead body burdens has not been found in any single prior study. This association is reasonable as will be seen from the ensuing discussion of individual lead exposures.

Large differences in blood lead levels by exposure to automobile traffic indicated a strong relation between the two in this study (Table 18). This is confirmed in the result of the statistical analysis using the traffic volume of the recruitment area but not in the other two traffic volume representations. This discrepancy may be explained in two ways. First, in the urban setting of Charleston where streets are relatively close together, children are not only exposed to lead from automobiles on the street facing their home, but also lead from other streets as well. Second, the lack of a relationship may be a result of a poor distribution of data points along the other two traffic volume representations.

Supporting the first explanation is the variation in the level of significance of the traffic volume variables and the percent variation in blood leads explained. This variation was in the order of increasing geographic coverage. Recuitment area was the most significant traffic variable and the model using that variable explained the largest variation in blood leads; daily traffic on the facing street was the least significant and explained the least variation; daily traffic volume within a 76 m radius was intermediate in both criteria.

Supporting the second explanation is that a detailed examination of the traffic count data for traffic density on facing street, and for that within a 76 m radius, shows a clustering of data points at the low end. As a matter of fact, the median traffic exposure was at the minimum traffic count in both instances. This outcome reduced markedly the value of these two representations of traffic exposures, thus suggesting that residence in the recruitment area was a better indicator of traffic volume in this study. In other areas, the broad "traffic recruitment area" model might not be the most accurate description.

The relationship of blood lead levels to soil lead exposure certainly was not surprising. Fairey and Gray's (1970) work clearly implied that such a relationship might exist. Barltrop's *et al.* (1970) results, though not apparently internally consistent, may be explained by our data. In their first study, soil lead levels did not exhibit as broad a range of values as ours, while the preliminary information given for their second study indicates a range of exposure possibly even greater than ours. Therefore, a relationship probably does exist, although fairly elevated soil lead levels are needed for it to appear. Work by Sayre *et al.* (1974) may provide evidence for the mechanism of exposure from dust or soil; that is hand to mouth activity. Dust from the hands of children exposed to higher lead levels was found to have higher lead levels than dust from the hands of children not so exposed.

The limited paint lead data that can be used to evaluate the reasonableness of our results come from Reece *et al.* (1972). In that study, paint lead levels roughly comparable to ours were found; the authors stated that no relationship with blood lead was derived but did not report the data to allow the determination of whether a trend existed. It should be noted, however, that our study population was twice that of Reece *et al.*

Therefore, in the context of available information, our results are entirely reasonable. They pinpoint further research which needs to be undertaken. This research includes confirmation of the soil lead relationship, confirmation of the paint lead relationship, and the elucidation of the relevant traffic exposure variable for children.

Confirmation of the soil lead results is needed both in different races and different geographic locations. These studies need a larger sample size, measurement of the lead contamination of the children's hands, and measurement of household dust, to be conducted in areas with low paint lead concentrations and low traffic densities. Possible study sites might include redevelopment areas in cities in which high soil levels may exist with low lead paint levels.

Although clinical impressions and lead screening programs have indicated that 1% weight lead paint is potentially hazardous, little effort has been made to quantify the relationship of paint lead levels with lead absorption. Our results suggest that only some of the painted household surfaces may be relevant. Efforts need to be undertaken to define more closely the relationship of paint lead level with blood lead and to determine which surfaces are really hazardous.

Our results certainly suggest that exposure to automobile traffic is an important determinant of children's lead burden; they also raise the question as to what is the best estimate of that exposure. In this study, soil lead exposure was the best index to environmental lead. Studies of multiple sources of lead also need to be conducted using the refined exposure estimates described above to enable scientists to clarify the relative contribution of lead from soil, paint, and traffic exhaust to children's lead burdens in different areas. It is clear, however, that failure to consider traffic volume and soil lead as well as lead paint in studies of human burdens could easily cause incorrect inferences regarding the environmental sources of nondietary lead intake.

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REFERENCES

Barltrop, D., Strehlow, C. D., Thorton, I. and Webb, J. S. 1974. Significance of high soil lead concentrations for childhood lead burdens. Environmental Health Perspectives. Experimental Issue 7:75-82.

Caprio, R. J., Margulis, H. L. and Joselow, M. M. 1974. Lead absorption in children and its relationship to urban traffic densities. Archives Environmental Health 28:195-197.

Daines, R. H., Smith, D. W., Feliciano, A. and Tracet, J. R. 1972. Air levels of lead inside and outside of homes. Industrial Medicine and Surgery 41:26-28.

Fairey, F. S. and Gray, J. W. III. 1970. Soil lead and pediatric lead poisoning in Charleston, S.C. Journal South Carolina Medical Association 66:79-82.

Gadsden, R. H. and Cannon, A. 1964. Measurement of erythrocyte G6PD activity. In Hemoglobin, Its Precursors and Metabolites, eds. Sundeman and Sundeman, pp. 332-336. New York: Lippincott.

Goldsmith, J. R. and Hexter, A. C. 1967. Respiratory exposure to lead: epidemiological and experimental dose-response relationships. Science 158:132-134.

Hammer, D. I., Finklea, J. F., Hendricks, R. H., Hinners, T. A. and Riggan, W. B. 1972. Trace metals in human hair as a simple epidemiologic monitor of environmental exposure. In Trace Substances in Environmental Health V., ed. D. D. Hemphill, pp. 25-38. University of Missouri, Columbia.

Knelson, J. H., Johnson, R. L., Coulston, F., Goldberg, L. and Griffin, T. 1973. Kinetics of respiratory lead uptake in humans. In Proceedings International Symposium Environmental Health Aspects of Lead, ed. D. Barth, A. Berlin, R. Engel, P. Recht, and J. Smeets, pp. 391-401. Luxembourg: Commission of European Communities.

Laurer, G. R., Albert, R. E., Kneip, T. J., Pasternack, B., Strehlow, C., Nelson, N. and Kent, F. S. 1973. The distribution of lead paint in New York City tenement buildings. American Journal Public Health 63:163-168.

Lin-Fu, J. S. 1972. Undue absorption of lead among children—a new look at an old problem. New England Journal of Medicine 286:702-710.

National Academy of Sciences. 1972. Lead: airborne lead in perspective. Washington, D.C.: National Academy of Sciences.

Reece, R. M., Reed, J. A., Clark, C. S., Angoff, R., Casey, K. R., Challop, R. S. and McCabe, E. A. 1972. Elevated blood lead levels and the *in situ* analysis of wall paint by x-ray fluorimetry. American Journal Diseases of Children 124:500-502.

Simpson, J. M., Clark, J. L., Challop, R. S. and McCabe, E. A. 1973. Elevated blood lead levels in children—a 27 city neighborhood survey. Health Service Reports 88:419-422.

Thomas, H. V., Milmore, B. K., Heidbreder, G. A. and Kogen, B. A. 1967. Blood lead of persons living near freeways. Archives Environmental Health 15:695-702.